A STUDY OF 100 CASES OF DEATH DUE TO ORGANO PHOSPHORUS COMPOUND POISONING

Dissertation

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INTRODUCTION

The desired activity of pesticides is to kill unwanted organisms such as insects, fungi, weeds and rodents. They comprise of a heterogeneous groups of substances which defy easy classification. A common and useful classification is by these target organisms into insecticides , fungicides, herbicides, molusciudies and rodenticides; in each group they are often subdivided by chemical group. Some chemicals have more than one type of pesticidal activity and may therefore appear in more than one category. For example, carbamates and organophosphates (OPs) includes insecticides, herbicides and fumigants and rodenticides. From a regulatory point of view, pesticides are often divided into agricultural (including horticultural) and non-agricultural types, the later including wood preservers, anti-jouling materials for boats, and insecticides used in public hygiene.

In Sri Lanka many thousand of Hospital admission each year due to AGROCHEMICAL POSONING (16649 IN 1983) with over a thousand deaths annually (1521 in 1983), of these about three

quarter were self administered, the remainder being accident & occupational)

Organo phosphorus compounds are extensively used as pesticides for soft bodied insects in agriculture. They have been imported in India Since 1951, but very few knew about the nature of these compounds as a virulent poison till the real food Poisoning Tragedy in 1958. This tragedy which took a toll of about hundred and odd lives, due to inadvertent stocking of food stuff (Wheat, flour, sugar etc) & Folidol (Parathion) packages in the same hold where the Folidol containers leaked and contaminated the gunny bags containing food stuff. Their easy availability & quick actions the reason for their popularity for suicidal & homicidal purpose.

The main dangers from agricultural chemicals lie in the pesticides, especially ORGANO PHOSPHORUS COMPOUNDS such as Paration Herbicides such as PARAQUAT, MONOOROLOPHES used in huge quantities through out the world and these there substances cause thousand of death in Southern Asia, Africa, and other developing countries.

REVIEW OF LITERATURE

HUMAN EXPOSURE TO PESTICIDES.

Exposure of human to pesticides can occur in a number of ways.

Occupational Exposure :

Farmers or farm workers and gardeners may be exposed to agricultural or horticultural pesticides during application of these pesticides on crops (Operator exposure). Similarly, operators are exposed to pesticides during non-agricultural application, as are in individuals treating stock animals. The most important routes of exposure in operators are the skin and the respiratory tract. Risk assessment is carried out by comparing calculated or actual observed operators exposure to the pesticides with the acceptable operator exposure level (AOEL) derived from animal or more rarely, human experimented data. The various rates of exposure may be considered either in isolation or in combination, when assessing the potential effects of pesticides in humans. Basic principles suggest the latest

approach in appropriate when considering exposure to pesticides with similar toxicological actions – a procedures suggested by the US Food Quality Protection Act (USA 1996). Biomarkers of exposure, where available, are used to assess exposures of the pesticide (eg: The Alkyl phosphates for organophosphates) but electro physiological biomarkers have been suggested in certain instances (Desi & Nagymajtency 1999).

CONSUMER EXPOSURE.

The general population may be exposed to pesticides from various sources but, in contrast to pesticide operators, ingestion is the most important route of exposure of the public. For as long as pesticides are required to help produce the volumes and quality of Food demanded by the public, exposure to residues of pesticides via the food chain is inevitable, however small. There is the added potential for exposure of the whole population through drinking water. Residue in food and water are the sources of pesticides that

attract greatest public concern. The fact that many of the public also use pesticides in their gardens and to treat their pets seems of lesser concern. In addition, they may be exposed, or during use in public hygiene or vector control also. Consumer pesticide exposure is compared with the acceptable daily intake (ADI), a level of exposure considered safe over a lifetime and derived, like the SOEL, from experimental toxicology data. Residue analysis is generally carried out on bulked samples of fruits and vegetables even when it is common practice to eat items in their entirety, recently it has become apparent that such bulking of samples may conceal considerable individual variation in pesticide concentrations in individual items (Harris 2000, Marks 2000).

POISONING:

However good systems for regulating pesticide accidents we are practice misuse of pesticides by a tine minority is inevitable. Not only have these agents been used to kill large mammals, birds of

prey, gams and fish, the more toxic of them have been used for suicide and murders. (Booke 1998).

ACCIDENTAL POISONING

Acute pesticide poisoning – defined as thikness attributes to exposure to excessive quantities of these chemical agents, resulting from contaminating of good chain seems to lie extremely rare, particularly when the pesticide has been applied in accordance with approved use. Only very occasional outbreaks are reported (Chandry etal 1998). This may reflect rue raity by it is conceivable that sporadic eases of pesticide poisoning with minor clinical symptoms are ascribed to microbiological course. Clinical poisoning by pesticides residues has been reported from

1. Spillage of pesticides on to food during storage or transport.
2. Eating grain or seed potaters treated with pesticides, where the commodity was not intended to human consumption.

3. Improper application of pesticides.

4. Failure to observe recommended intervals between the last pesticide application and harvest.

The pesticides involved have generally been ones with high acute toxicity (LT) <20mg/Kg body weight, such as the insecticides Endrin Parathion and Oldicarb or Rodenticides such as Thallium Sulpate and Sodium, Fluoride, other pesticides that have produced morbidity by ingestion with food include Organic mercury fungicides (Ferrer & Lobral 1991) when adverse effect of pesticides occur in occupational settings, which seems to be more common, the connection between cause and effect is easier to make occasionally the outcome is fatal (Wesseling et all 1997).

DELIBERATE POISONING

On a worldwide basis, pesticides are alleged, probably correctly on to be responsible for hundreds and thousands of cases of acute poisoning and may thousands of deaths each year. The sad fact is

that the vast majority of them are the result of deliberate self poisoning rather than accidents. The oral route is the usual one, when pesticides are consumed with this specific objective, rarely the agent may be injected. The pesticides involved are inevitably those that are most readily accessible (eg: OP and carbamate insecticides in Sri Lanka, and metal Phosphide Rodenticides in India) or have established a reputation for having a fatal outcome. (eg: the herbicide, paraquat).

DIAGNOSIS

Fortunately, most individuals poisoned with pesticides are not so seriously unwell, that they cannot give a history of exposure, provided they know they have been exposed and are willing to durilge this information, occasionally religion, cultural background and guilt are inhibiting factors. Diagnosis of pesticide poisoning in the absence of a history volunteered by the victim or strong circumstantial evidence suggesting the possibility require a high

index of clinical suspicion, Clinical features are seldom diagnostic and pathognominic though Physicians in countries where OP and Carbanote poisoning is common may have the experience to identify the acute choleniregie illness produced by them even in the absence of a history. Other documented scenarios include a bleeding diathesis prolongation of prothrombin time, surreptitris injection of anticoagulants possibly in the foum of rodenticides should be suspected. Similarly, an acute gastrointestinal upset followed after 2-3 days by evolution of oropharyngeal burns, renal impairement and the onset of respiratory symptoms would suggest paraquat poisoning. The possibility of poisoning through food or water will be suspected, soon or later when a number of individuals present to hospital emergency rooms within a short period with similar unexplained illness.

One of the benefits of a prior approval method of pesticide regulation is that analytical methods for estimation of concentrations of active ingredients (usually in blood, sometimes in urine) are some times available either from the company marketing the pesticide or

from the government regulatory body, however they are not available in hospitals or on emergency basis. In other cases, metabolites may have to be estimated to confirm a diagnosis of poisoning (eg. Alpha napthol in suspected exposure to carbonyl) Although hospital laboratories are able to perform specific test to identify the presence of only one pesticide (paraquet) they are usually able to perform diagnostic tests indicative of poisoning with others, such as reduced plasma and red blood cells cholinesterase activity in OP and Carbamate poisoning, or the prolongation of prothrombin time with over dose of anticoagulant rodenticides.

PRINCIPLES OF TREATMENT OF PESTICIDE POISONING.

The care of individuals poisoned with pesticides differ little form that of patients with other illness, particularly other forms of poisoning. All require supportive care and observation commensurate with their clinical condition, the objective being to ensure, as far as possible, the effective delivery of oxygen and its

utilisation by tissues for as long as is necessary for the toxin to be eliminated from the body. The main components of this care are directed at maintaining a clear air way and adquate ventilation, producing an effective cardiac output, and correcting potentially life threatening metobolic abnormalities.

The additional therupeutic interventions specific to poisoned patients that must be considered are the need for decontamination (gastro intestinal tract, skin or eye, as appropriate) antidotes and the use of techniques intended to enhance the rate of natural elimination of pesticide that has bee absorbed.

But decontamination is the term currently use to encompass treatments intended to reduce absorption of poisons from gastrointestinal tract. Over the decades many treatment methods have been used but recently critical assessment of their value has failed to satisfy the present day requirements for evidence induction of vomiting (whether by syrup of ipeconcuanha salt solutions or other

means) and the use of cathartics have been largely consighed to history. Even the indication for gasture lavage and to circumstance in which a Toxicologically significant quantity of a substance has been ingested in the intervention can be implemented in an hour of ingestion. clearly, few poisonings are likely to fulfill these criteria .

Antidotes are available for use in sever poisoning with metal containing pesticides, OP and Carbamate insecticides and anticougulant rodenticides. Forced diureses in such poisoning is of no value and is potentially life threatening. Dialysis (Peritoneal / hemo) is more likely to be indicated for toxin induced renal impairment than for removal of pesticides from the blood. Similarly there is no obvious role for hemoperfusion though niches for plasma pheresis (Chlorate poisoning) and alkalinzation of the urine (poisoning with chlorophenosy herbicides) may arise occasionally. These are discussed under the appropriate pesticides.

INSECTICIDES :

Many insecticides effect nervous system of both insects mammals, achieving selectively as described above, while the newer ones exploit their ability to disrupt features specific to insects. The toxicity of the former group tends to be manifested in the central nerves system (CNS) and or perepheral nervous system, whereas the toxicity of the latter tends to be less specific. Consequently insecticide may be logically classified as neurotoxic and non neurotoxic. Other classifications are also used particularly that which divides insecticides into those of natural origin (eg. phrethris) and there that are synthetic. The neurotoxic group contains both natural & synthetic types.

ANTICHOLINESTERASES

The groups of anticholinesterases, the organophosphates and the carbonates are widely used as agricultural

insecticides. Marrs 1996, Moretto 1998, Marrs 2001). One OP, malathion, is used in human medicine to treat head lice and another, metri-fonate (trichlorfon), in schistosomiasis (Aden-Abdi et al 1996). Organophosphates also have an important role in veterinary medicine as dips to control sheep scab and fly strike, and to kill warble fly in cattle. Anticholinesterases are also the active components of various proprietary treatments for fleas in cats and dogs. Anticholinesterase carbamates also find a role in human medicine, e.g. pyridostigmine in myasthenia gravis. Pyridostigmine has also been studied as a prophylaxis for OP nerve-agent poisoning, specially soman.

The anticholinesterases are often more acutely toxic than OC insecticides and some, e.g. mevinphos, are particularly toxic. In contrast others, especially the phosphorothioates with a P=S bond (see below), are of low acute mammalian toxicity.

The action underpinning the acute toxicity of OPs and carbamates is inhibition of cholinesterase activity bought about by phosphorylation or carbamylation of the enzyme respectively. Acetylcholinesterase hydrolyses acetylcholine in the nervous System

and is also present in red blood cells. When inhibited, the neurotransmitter acetylcholine accumulates at parasympathetic effectors sites, synapses, neuromuscular junctions and in the CNS. resulting in what is often referred to as the "acute cholinergic Syndrome." But yrylcholinestcrase (also known as non-specific cholinesterase or pseudocholinesterase) has a more obscure role and is notably present in the plasma.

AGING:

With some OPs a further retain, known as aging, has to be considered. Research on the treatment of poisoning with the nerve agent, some one found that reactivation of the inhibited enzyme failed to occurs with oxime deactivators unless they were given in very short time of exposure. Aging appears to consist of monodealkylation to leave monoalkylphosphory enzyme which reactivates neither spontaneously nor with enzyme reactivates. Aging half lives for dimethoxy phosphorylated cholinesterases are in the region of 2-9 hrs, so that, in clinical practice aging should not be

a problem providing treatment with an antidote in instituted promptly.

THE MECHANISM OF CHOLINESTERASE INHIBITION

Inactivation of cholinesterases by OPs results when the pesticide reacts with a serine residue on the enzyme, resulting in the leaving group of the OP (see Figure below) being lost and a dialkyl-fosphory derivative of the enzyme produced. Since most insecticides contain either two methyl or two ethyl R groups, the product is, more accurately,a

$$\begin{array}{c} R^1 \\ | \\ O \\ | \\ R^2\text{—}O\text{—}P{=}{=}O(S) \\ | \\ X \end{array}$$

dimethoxyphosphorylated enzyme or a diethoxyphosphorylated enzyme. Quantitative differences in the acute toxicity produced by OPs are partly the result of varying rates of formation of the OP-acetylcholinesterase complex, or hydrolysis of this complex, and of

the aging reaction (see below) as well as absorption, distribution and metabolism.

Reactivation

Inhibited cholinesterase may undergo spontaneous reactivation, the kinetics of which are independent of the structure of the leaving group of the OF. Reactivation of dimethoxyphosphorylated enzyme occurs within a few hours and is considerably quicker than that of the diethoxy equivalent. Similarly, complexes containing one alkylthio group and one alkoxy group reactivate faster than those containing two alkoxy groups. Spontaneous reactivation of complexes containing larger R groups, e.g. isopropoxy and disecbutoxy, is slow or non-existent (WHO 1986, Wilson et al 1992, Mason et al 1993, Moretto 1998). The stability of phosphorylated enzyme is generally greater than that of carbamy- lated enzyme so that carbamate poisoning is of shorter duration, The rate of reactivation of OP-inhibitcd cholinesterase may be increased by the use ofoximes (cholinesterase reactivators).

Acute anticholinesterase poisoning - the "cholinergic syndrome"

FEATURES

The acute cholinergic syndrome produced by the OPs and carbamaces is similar. The consequences of parasympathetic enzyme inhibition include bronchorrhea, bronchial constriction, hypersalivation, constriction of the pupil of the eye with ocular pain and dimming of the vision, abdominal colic, and involuntary micturition and defecation. Accumulation of acetylcholine at neuromuscular junctions results in muscle fasciculation and or increasing muscle weakness. CNS effects may include confusion and apprehension, with convulsions occurring in severe poisoning. The effect on heart rate is unpredictable, and either tachycardia or bradycardia may occur. Arrhythmias, including torsade de pointes, are seen in severe poisonings (Saadeh et al 1997). Respiratory paralysis, which may be of central and/or peripheral origin, is the main cause of a fatal outcome although cardiac effects may contribute (Zwiener & Ginsburg 1988, Tsao et al1990).

MANAGEMENT

Intensive supportive measures form the mainstay of the management of acute anticholinesterase poisoning, whether caused by OPs or carbamates. Antidotes may also be invaluable. Atropine, often needed in exceptionally large doses, counteracts the parasympathetic consequences of poisoning by both OPs and carbamates. Convulsions and muscle fasciculation can be controlled with diazepam (Johnson & Vale 1992). Cholinesterase reactivators such as the oximes, pralidoxime chloride (2-PAM) or obidoxime, improve muscle power (Bismuth et al 1993, Szinicz et al 1996) and are indicated in severe poisoning with OPs, but not carbamates (see below).

Chronic effects on the nervous system

It is generally believed that, provided the patient survives, the symptoms and clinical signs of anticholinesterase poisoning are completely reversible. However, survival from high doses of OPs and carbamates may result in long-term clinical and electrophysiological changes in the nervous system. The life-threatening features of acute poisoning (see above) are potential causes of anoxia, to which the

CNS is especially vulnerable- It. is thus biologically plausible that major intoxication is sometimes associated with residual CNS changes (Holmes & Gaon 1956, Durham et al 1965. Tabershaw & Cooper 1966, Burchfield et at 1976, Korshak & Sato 1977, Bartels & Friedel 1979, Dufly et al 1979, Hirshberg & Lei-man 1984, Savage et al 1988). More debatable is whether longterm, low-dose exposure without hypoxia produces chronic effects, and it is important that the two scenarios are not confused. Studies have been carried out specifically on individuals exposed long-term to low doses, usually in occupational contexts (Ames et al 1995, Beach ec al 1995, Stephens et al 1995, Fiedler et al 1997, Institute of Occupational Medicine 1999), and reviews (Institute for Environment and Health 1998, Royal College of Physicians of London and Royal College of Psychiatrists 1998, European Centre for Ecotoxicology and Toxicology of Chemicals 1998, Committee on Toxicity 1999) have concluded that the evidence for adverse outcomes was less consistent after long-term, low-dose exposure than after acute poisoning with OPs, although some studies had shown effects.

Diagnosis

Laboratory measurement of cholinesterase activity in plasma and/or red cells can be used in addition to the characteristic clinical findings to confirm a diagnosis of acute antichohnesterase poisoning. The numerous esterases in the body differ in their sensitivity to

inhibitors, and butyrylcholinesterase is usually the cholinesterase most susceptible to inhibition. It also reactivates relatively slowly, particularly with diethoxy pesticides (Skrinjaric- Spoljar et al 1973, Worek et al 1999, Mason et al 2000), making it a useful marker of exposure to anticholinesterases. In contrast, erythrocyte acetylcholinesterase inhibition often correlates better with cholinergic status but reactivation can take place sufficiently quickly that interpretation of measurements of cholinesteraseaictivity in blood requires particular care (FAO/WH0. Mason et al 1,993), particularly as reactivation can occur ex vivo in blood samples. All the acetylcholinesterases in those organisams that have been studied arc the same gene product, and therefore can be expected to have the same characteristics of inhibition and reactivation. However, concentrations of inhibitor may be dissimilar at different sites in the body. The CNS, in particular is to a greater or lesser degree protected from circulating toxicant the blood-brain barrier so that even red cell acetyl cholinesterase activity may correlate poorly with clinical status.

Concentrations of alkylphosphates, metabolites organo phosphates, can be measured in urine but the excretory pattern varies according to the particular OP and measurements are more useful for industrial monitoring than diagnosing poisoning. Blood and urine levels of parent compound can sometimes be measured

Organophosphates

CHEMISTRY

Organophosphate pesticides are esters of phosphoric, phosphonic or phosphorothioic or related acids with the general formula shown in Figure with, OP insecticides. R' and R2 are usually either both methyl groups or ethyl groups, while the X or leaving group can be any one of a large variety of moieties. Rather than having a P=0 group, many pesticideal OPs P=S group (i.e. they are phosphorothioates) and tend to be of lower acute mammalian toxicity than their corresponding phosphates and phosphonaces (their oxons") Phosphorothioates only acquire toxicity after conversion of their P==S moiety to a P==0 moiety (WHO 1986). A notable

example is malathion (FAO/WHO 1998). Organophosphate chemical warfare nerve agents are often phosphonofluoridates and contain more bulky alkyl groups.

ACUTE POISONING

Acute poisoning with OPs results in the "cholinergic syndrome" described above. Additional features include hyperamylasema which is not uncommon (Lee et al 1998) and acute pancreatitis which is (Panieri et al 1997).

INTERMEDIATE SYNDROME (IS)

Senanayake & Karalliedde (1987) reported a syndrome that they named the "intermediate syndrome" because it occurred after the acute cholinergic syndrome and before delayed polyneuropathy. The syndrome does not occur with carbamate poisoning and is probably the same as the "type II syndrome" described by Wadia et al (1987). Numerous cases have been recorded, for instance that by Karademir et al (1990). The cardinal feature is proximal limb paralysis starting up to about four days after poisoning. This may be

the result of the myopathy observed post mortem in cases of human poisoning (de Rueck & Willems 1975 and in experimental human animals (Presusser 1967,Wecker et al 1978), and which appears to be initiated by accumulation of calcium in the region of the motor end plate (Inns et al 1990). The major alternative etiological hypothesis is that of depolarization blockade secondary to secondary to inadequate treatment with cholinesterase reactivators. In addition to supportive treatment, therefore, further doses of oximes should be given. Respiratory muscle weakness frequently necessitates the use of assisted ventilation.

ORGANOPHOSPHATE – INDUCED DELAYED POLYNEUROPATHY (OPIDP)

OPIDP is a symmetrical, sensory and motor axonopathy that tends to be most sever in long axons and occurs 7-14 days after exposure (He et al 1998). It is a polyferation (Bouldin & Cavanagh 1979a, b Cavanagh 1982) accompanied by changes in the spinal cord and medulla oblongata (Barrett et al 1985). Delayed polyneuropathy is specific to OP poisoning and does not occur in carbamate poisoning. In severe cases, the legs may be completely paralyzed while less severely affected individuals exhibit the characteristic high stepping gait associated with foot drop. Some recovery of the peripheral component may occur but the central component of OPIDP appears not to reverse (Vasilescu & Florescu 1980). No specific treatment exists (Barrett et al 1985). In particular, the antidotes for treatment of acute poisoning are ineffective.

The molecular basis of OPIDP is not fully understood but the syndrome is accompanied by and may be caused by, inhibition of neuropathy target esterase (NTE), an esterase present in nervous

tissue. Inhibited NTE may then undergo aging similar to OP >75% is productive of development of OPIDP 10-20 days later (Moretto 1998). Structure / activity requirements for inhibition of acetyl cholinesterase and NTE are quite different as is demonstrated by the f act that many powerful anticholinesterase OPs are unable to cause OPIDP while the most famous outbreak (Ginger jake paralyis due to contamination of illicit alcohol during the period of prohibition in the USA) was caused by tri-ortho-cresyl phosphate, a weak cholinesterase inhibitor (Woolf 1995)

Hens are very susceptible to OPIDP and (el-Sabae et ak 1981, Gallow & lawryk 1991)

OTHER EFFECTS:

Organophosphates may have other toxicological effects including genicity and carcinogenicity as well as specific toxicity directed at the heart, kidney and other organs (Singer et al 1987, Baskin Whitmer 1992, Pimentel & Carrington da Costa 1992, Wedin 1992). Some of the effects observed with individual OPs may be independent of their easterase-inhibiting properties.)

Atropine •

Mode of action : Blocks the muscarinic manifestations of organophosphates. However, since. atropine affects only the postsynaptic muscarinic receptors, it has no effect on muscle weakness or paralysis.

Dose: 1 to 2 mg i.v or i.m (adult) : 0.05 mg / kg i.v .(child); every 15 minutes until the end point Is reached i.e., drying up of tracheobronchial secretions. Pupillary dilatation and tachycardia are not reliable indicators of the end point. Once the end point has been reached, the dose should be adjusted to maintain the effect for at least 24 hours.'

Availability : Atropa inj (Jawa Pharma) of 0.6 mg/ml, 2 ml ampoules; TrpJaine inj (Neon Labs) of 0.6 mg/ ml, 1 ml ampoules.

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Pralidoxime (Pyridine-2-aldoxime methiodide ; 2-PAM)

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Mode of action : It is usually given along with atropine. Pralidoxine competes for the phosphate moiety of the organophosphorus compound and releases it from the acetylcholinesterase enzyme thereby liberating the tatter and

reactivating it. While it is advisable to begin pralidoxime therapy' within 48 hours of poisoning, it can be administered even much later with beneficial effects.[2]

Dose: For adults – 1 to 2 gm in 100 to 150 ml of 0.9% sodium chloride, given i.v. over 30 minutes. This can be repeated after 1 hour and subsequently every 6 to 12 hours, for 24 to 48 hours. Maximum dose should not exceed 12 gm in a 24 hour period

For children - 20 to 40 mg/kg to a maximum of 1 gm/dose given i.v, and repeated every 6 to 12 hours for 24 to 48 hours.

It is generally not advised for the treatment of carbamate overdose, especially carbaryl.

Availability : PAM tabs, 500 gms, for oral use (Shree Ganesh) PAM-A Korea inj of 500 mg in 20 ml for i.v. use (Shree Ganesh) : PAM-A inj of 500 ml, for i.v. use (Panacea)

Diazepam

Some studies indicate that the additional of diazepam to atropine and 2 – PAM improves survival 3 it reduces the risk of seizure – induced brain and cardiac damage.

Dose: For adults – 5 to 10 mg i.v. slowly, every 15 minutes, upto a maximum of 30 mg.

For children – 0-25 to 0.4 mg/kgi.v. slowly, every 5 to 10 minutes, upto maximum of 10 mg.

HISTORICAL ASPECTS

Prior to second world war, only the reversible anticholinesterase agents were known, of which physostigmine is outstanding example. Shortly before and during the second world war, a comparatively new class of highly toxic chemicals the organophospates were developed chiefly by schrader, of I.G. Farben industries first as agricultural insecticides and later as potential chemical warefare agents.

In highly potent compound of organophosphorous anticholinesterase series, Tetracthyl pyrophosphate as published by clermont in 1854.

During synthesis and investigation of approximately 2000 compounds, Schrader (1952) defined structural requirements for insecticidal activity. Amount this, parathion became the most widely employed insecticide. prior to and during second world war, the efforts of Scherader's group were directed towards the development of chemical warfare agents. The synthesis of several compounds of

much greater toxicity than parathion such as sarin, soman and tabun resulted but were kept secret by the German goverment.

In the 1950's a series of heterocyclic aromatic and napthyl carbamates were synthesized and found to have a high degree of selective toxicity against insects and to be potent antiche agents (Glysin 1954). Among currently employed insecticides are i-napthyl No-Methyl carbamate (carbaryl Sevin) and 2- Isoprophil poxyphenol-N, methyul carbamate (Baygon) (Fukuto 1972, Murphy 1980)

SOME OF THE COMPOUNDS- CHEMISTRY – AND PROPRIETARY NAMES

ALKYL PHOSPHATES.

Compound	Chemical Name	Proprietory Name
HETP	Hexaethyl Tetraphosphate	Teran Fesover
TEPP	TETRACTHYL PYROPHOSPHATE	
OMPA	Octa Methyl Pyrophophoramide	Schraden
DIME FOX	Bis (Dimethyl Amina Fluoro Phosphate Oxide)	
ISOPE STOX	Bis Isoprophyl Amino Fluorophosphine oxide	Pestox 15
MALATHION	S(1-2 Dicarboethoxy ethyl) o' Dimethyl Phosphonodithioate	Compound 4049 Kill Bug Cemexol
SULFOTEPP	Tetra ethyl. 0. Dithio pyrophosphate	Joothi OIl Bugsolin 20
SYSTOX DEMETON	0-0- Diethyl 0-2 Ethyl mercaptoethyl Thiono Phosphate.	Dithione ASP 47
DIPTREX	0-0 Dimethyl 2-2-2- Trichlorol-Hydroxy ethyl phosphonate	Tug on Batt.

ARYL PHOSPHATES

Compound	Chemical Name	Proprietory Name
PARAOXON	0-0 Diethyl O-P Ntro Phenyl Phosphate	R 600 Mintacol
PARATHION	0-0 Diethyl O-P Ntro Phenyl Thiophosphate or Diethyl Thio Phosphoric easter of P. Nitrophenol	Fillidol Edetox Kilkphos, Niran Rhyntox Orientlal bug Bait.
EPN-O	Ethyl O.P Nitrophenyl Thiophosphonate	E.P.N.-600
METHYL PARATHION	0-0 Diethyl -) (Isoprophl 4-Methyl drinidyl (6) Thisphosphate.	Tik – 20
4-METHYL UMBELLI PERONE	0-0 Diethyl Thio Phosphate	
DIMETHYL AMINO ETHOXY	Phosphoryl Gyanidate	Tabun
ISO Propoxy METHYL	Isoprophl Methil Phosphate Fluridate	Sarin

Good Man and Gil Man

The sign and symptoms of organophosphorous compounds are show in tables given below: (Vale et al)

MUSCARINIC MANIFESTATIONS

OF OGANOPHOSPHOROUS

COMPOUND POISONING

SYSTEM	EFFECTS FOLLOWING LOCAL EXPOSURE	EFFECTS FOLLOWING SYSTEMIC ABSORBTION
Cardiovascular		Bradycardia Hypotension Hear Block
Respiratory	Tightness in chest Wheezing	Tightness in chest Wheezing Dyspnosa Increased bronchial secretions. Cough Pulmonary Oedema Cyanosis.
Gastrointestinal		Anorexia Nusea Vomitting Diarrhoea Abdominal Cramps Tenesmus Faecal

		incontinance.
Conjunctiva	Hyperemia Miosis (Usually maximal sometimes unequal	Miosis
Cilliary body	Eye pain on Focusing Slight dimmness of vision Frontal headache	Blurring of vision
Salivary glands lacrimal glands Sweat glands		Increased salivation Increased Lacrimation Increased Sweating
Nasal mucous Membrance	Rhinorrhoea Hyperosemia	
Bladder		Frequency Involuntry incontinence.

NICOTINIC MANIFESTATIONS:

Striated musclel		Muscle Twitching Faciculation (Eye lids, Facae, Calf Pirmarily affectd)
Sympathetic ganglia		Pallor Tachycardia Elevation of blood pressure Hyperglycemia

CENTRAL NERVOUS SYSTEM AND MISCELLANEOUS MANIFESTATIONS:	
Central Nervous System	Giddiness, anxiety, restlessness, emotional liability, excessive dreaming insomnia, night mares, Headache, drowsiness and difficulty in concentrating. Confusion, dysarthria, ataxia, Muscle aches cramps EEG: Irregularities in rhythm variation and increase in potential and intermittent busta of abnormally slow waves of elevated voltage may be found
Liver	Acute increase in urinary excretion of urobilinogen.
Blood coagulation	Hypercoagulability with shotended prothrombin time. Increased prothrombin consumption. Increased factor VII. (All rate and not usually of clinical signifiance.)
Skin	Contact dermatitis (rare aphthous ulceration and stomatitis (rare)

Fatal Period :

The required oral and dermal Lethal dose values of various poisoning are given below:

(Bashyam eta al)

Name of Pesticides	**Oreal Mg/Kg**	**Dermal Mg/Kg.**
Diazion	300-600	500-1200
Dichlorvos	25-30	70-900
Dichrotophos	15-45	150-225
Dimethoate	200-300	700-1150
Fenthion	200	1300
Fensulpholthion	2-4	13-14
Formothion	400	40-600
Malathion	1400-1900	1000
Mythyl O-demeton	50-75	300-40
Methyl Parathion	3-6	4.200
Monocrotophos	13-23	122
Phorate	2-3	70-300
Phosphomidon	15	125

Phasolone	135	1500
Thimeton	100	200
Quinolphos	62	80
Vamidothion	64-600	11

COMMON EFFECTS OF ACUTE ORGANOPHOSPHORUS TOXICITY

Muscarinic	Nicotinic
Peripheral	
Gastrointestinoal	Neuroumuscular
Salivation	Muscular fasciculations/ twitching
Increased gastrointestinal motility	
Abdominal cramping/discomfort	Rigidity
Vomiting	Cramps
Diarrhea	Weakness
Fecal incontinence	Hyporeflexia
Ocular	Paralysis
Miosis	Hypoventilation
Blurred vision	Excessives ympathetic output (from simulation of post ganglionic sympathetic neurons and adrenal catecholamine release)
Lacrimation	
Respiratory	
Rhinorrhea	
Bronchorrhea	Mydriasis
Bronchospasm	Techycardia
Dyspnea	Hypertension
Coughing	
Hyperventilation	
Hypoxemia	
Cardiac	

Bradycardia/abradydysrhythmias

Cardiac conduction delays

Hypotension

Genitourinary

Urination

Urinary incontinence

Dermal

Sweating

Central nervous system

(Nicotinic central nervous

system receptors may also playa

small role)

Agitation

Confusion

Hallucinations

Somnolence

Ataxia

Coma

Respiratory depression

Seizures

MEDICO LEGAL IMPORTANCE OF POISONING

Poison is a substance sold, liquid or gaseous which introduced into or brought into contract with aliving bodyproduces ill health, disease or death. If such substance even if non-toxic is administered with intention of killing or causing injury to a person. The person who administer it is punishable under the law. (Narayana Reddy)

Poisoning may result either for criminal purposes eg., with intent to kill or causing injury (Momidical Purposes) or for stupefying to facilitate a crime eg. robbery or rape or to produce abortion. It may result as a suicidal attempt or accidental (Mallik).

As a routine the medicao-legal cases are autopsied with the requisition from the police or RDO for those succumbed to the effect of organo phosphrous compounds ingestion. Bits were taken from the body and sent for Chemical analysis.

AIM OF STUDY

The Aim of study is to make an objective assessment of cases of deaths due to OPC Poisoning in respect of the reasons for consuming OPC poisons, the duration of survival after consumption. Post-mortem findings, amount of absorption, age at which there is maximum incidence, religious predominance and analysis of the toxicological report to arrive at a comprehensive conclusion about this commonest type of agricultural insecticide which is used as a substance to commit suicide.

MATERIALS AND METHODS

Only such of those cases of Organo phosphors Poisoning brought to the causality department of Govt. General Hospital, Madras Medical College, and brought dead cases of same poisoning brought with police memo were in general & analysed with the available data in respect of postmortem findings their chemicals analysis report etc.

About 100 cases of Organo phosphorous compound poisoning were taken for consideration in this study.. In all cases are the documents submitted by the investigation officer for doing the postmortem were thoroughly pursued regarding the age sex, religion, socio economic study identification inquest form (Form 86) and after the postmortem examination was conducted in all the bodies and finding were noted.

Chemical analysis examination was done in all the 100 cases and report were obtained. The chemical analysis report were analysed to find out the level of absorption in each case and taken in to consideration.

In all the cases the period of hospital stayed for the time of consumption of poisoning till the time of death were noted and the reason for consumption were noted during the period of hospital stay the treatment given were also thoroughly analysed, even though this study of the treatment part is not taken in to consideration.

After perusing the above said dates a detailed master chart was prepared and each is subjected to statistical analysis

DISCUSSION

Of the hundred cases of death due to organo phosphorus compound poisoning taken for study, the cases were grouped under various age group categories for analytical purpose. According the cases were grouped as

(i) 0 to 10 Years

(ii) 11 to 20 Years

(iii) 21 to 30 Years

(iv) 31 to 40 Years

(v) 41 to 50 Years

(vi) 51 to 60 Years

(vii) 61 to 70 Years

(vii) About 70 Years

Among the 100 cases, under the category of 0-10 Years only one case is reported. And that single female child (4 years) also falls under the peculiar manner of death namely homicide by mother by way of intramuscular injection of malathion. Under the second sub-

clause of 11 to 20 years 17 cases were reported. Among the 17 cases 11 cases were male & 6 female. Abdominal pain is cited as the reason for consumption of OPC in 10 cases. Of the remaining 7 cases 2 cases were due to financial crisis, 2 cases were due to depression caused by parental scolding, one case was due to dowry harassment and two more cases were accidental poisoning.

Under the age group of 21 to 30 years 31 cases were reported. Among the 31 cases 21 cases were male and 10 cases were female. The reason for consumption of poisoning under this sub clause falls under various headings, 18 cases were said to be due to abdominal pain and 8 cases of depression, one accidental exposes of agricultural spray, one case of love-failure, 2 cases of unemployment and 1 case of adultery.

Under the age group of 31 to 40 years, 18 cases were reported. Among the 18 cases, 14 case were male and 4 cases were female. In this category one case is said to be due to HIV infection, of the remaining cases, 11 cases were due abdominal pain, 3 cases of financial crisis, one case of drowning one case of chronic skin disease and case of dowry harassment.

Under the age group 41 to 50 years, 18 cases were reported. In this sub-class 17 cases were male and one female case. In this group 12 cases of death due to abdominal pain, one case of chronic alcoholic ending up with intractable depression, one case of family quarrelanural. Two cases of financial crisis and two cases of psychological depression.

Under the age group of 51 to 60 years, 5 cases were reported. Among these 4 were male and one female. Out of the 5 cases in this group one case is due to abdominal pain, two cases of depression, one case of financial crisis and one case of family quarrel

Under the age group of 61 to 70 years only two cases were reported. And those two cases were male and both cases were due to depression.

Only one case is reported under above 70 years age group, which is a female case and the reason for consumption of OPC is depression.

Among the group of subjects studied above the maximum incidence of OPC poisoning is in the group of 21 to 30 years (31

cases). Next to this category, high incidence is reported under 31 to 40 and 41 to 51 years (18 cases in each group) Under the age group of 11 to 20 years 17 cases were reported.

Out of 100 cases studied 74% of cases were male remaining were female.

Nearly 80% of cases of OPC poisoning falls under middle age group ie. between 11 to 50 years the remaining cases falls under extreme age groups.

Abdominal pain has been cited as a reason in majority of cases as per police record (History of the case given by the Police)

The other common reason for consumption appears to be psychological depression and financial crisis.

The maximum number of cases suicidal in nature, Accidental and homicidal death were negligible.

Based on the socio-economic status of the individual, the highest incidence of OPC poisoning is reported in the low socio-economic category and people of below poverty line.

CONCLUSION

Out of the 100 cases of death due to OPC poisoning taken up for study, there is a clear male preponderance, which accounts for nearly 3 cases among every four cases. This is because of the head of the family in every house is invariable a male person and he is the one who faces the brunt of the crisis.

Even though the abdominal pain is cited as the reason in majority of the cases, the psycho-social problem, financial crisis were the main reason for committing suicide among the general population.

Majority of case belong to the low socio-economic group of people who work as farmers are as contract labourers in agricultural Sector.activities. Since OPC products are available as over the counter product (OTC) at a cheap cost, accessibility and availability of the said product is easy for people who belong to the lower middle class and people of low Socio-economic status. The responsibility for an individual is very high only during the middle age i.e between 20 to 50 years. Inability to face the challenges in life during this period makes an individual more vulnerable to suicidal tendencies,

which results in ending his life to get rid of the problems & responsibilities.

To sum up, due to the reasons and explanations above said makes organophospherous compound poisoning one of the major killer disease among the people of low socio-economic life style.

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
1.	BD	1/4/88/3 4-10-03 Dr.VS	NityanandaMa	25	M	UMA	H	AbdoMainal Paid	NK	-	3-10-03	NK	Cyanosis of nail beds All organs congisted S: Contain 100 Mal of Fluid with kerosine odor with subMaucosul heMaorrhage	2005/03 dt;10-11-03	S; I L&K BR BL	Deducted OPC ND ND ND
2.	622464	1495/03 6-10-03 Dr.R.S	Nataraj	42	M	MAa	H	AbdoMainal pain	5-10-03 10 PMA	5-10-03 11.30 pMa.	6-10-03.3.10 aMa	5 Hrs. 10 MAts	Cyanosis of nail beds All organs congisted S: Contain 180 Mal of Fluid with kerosine odor with	2192/3 Dt.28-11-03	S: I L & K BR BL	Deducted OPC
3.	622375	1503/03 7-10-03 Dr.MA.S.	JayaraMaan	52	M	MAa	H	AbroMainal Pains	4-10-03 7 pMa.	4-10-03 8.30 p.Ma	6-10-03 11.40 pMa.	2 days 4 Hr. 40 MAt	Cyanosis of nail beds All organs congested contain 150 MAl of Yellow colour fulid with Kerosine odore	1896/3 dt. 20-10-03	S: I L & K BR BL	Detected OPC ND ND ND
4.	629127	1707/03 17-11-03 Dr.MA.S.	MAurugan	25	M	UMA	H	Occupational Agri. Spry	14-11-03 11 a.Ma.	15-11-03 at 10.20 aMa	16-11-03 9.30 aMa.	2 days 20 Mats.	Cyanosis of nail beds All organs congested S.EMapty subMaucosul heMaorrhage	2179/03 dt.30-12-03	S: I L & K BR BL	ND ND Detected OPC
5.	BD	1764/03 29-1-03 Dr. V.S.MA	ShanMaugaMa	32	M	MAa	H	AbdoMainal Pain	N K	-	28-11-03 at 12.30 pMa.	NK	Cyanosis of nail beds All organs congested contain stoMaach contains digested food particals withKerosine	2278/3 dt.20-01-04	S: I L & K BR BL	Detected OPC ND ND ND

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
													odore with su			
6	626293	1631/3 dt 4-11-03 Dr.MA.S.	MAurugavel	29	M	UMA	H	Depression	29-10-03 NK	29-10-03 7.11 pMa.	3-11-03 5.pMa	3 days 10 Hrs	Cyanosis of nail beds All organs congested contain stoMaach contains 100 Mal Yellow colur fluid no specific odour with subMaucosul heMaorrhage	2078/03 17-11-03	S: I L & K BR BL	Detected OPC ND ND ND
7.	62689	1632/03 dt.4.1..03 Dr.MA.S.	Kesavalu Raju	30	M	MAa	H	Depression	1-11-03 at 6 aMa	1-1-03 6.35 pMa.	3-11-03 6.45 aMa	2 days 45 Mats.	Cyanosis of nail beds All organs congested contain stoMaach contains 100 Mal Yellow colur fluid no specific odour	NTPT/ TOX 83/3 dt.2-1-04 RFSL Tirupathi	S: I L & K BR BL	Detected OPC
8.	637122	36/04 dt. 8-1-04 Dr. A.P.	Devadoss	42	M	MAa	H	Depression ((Alcoholic)	4-1-04 at 2 pMa	4-1-04 5.30 p.Ma.	7-1-04 9 aMa	2 days 9 hrs	Cyanosis of nail beds All organs congested contain stoMaach contains 150Mal blackish brown fluid no specific odour with subMaucosul heMaorrhage	79/04 dt. 13-2-04	S: I L & K BR BL	Detected OPC
9	B.D.	262/04 23-2-04 Dr.G.P.	Velu	45	M	MA	H	Depression	21-2-04 11.00 pMa.	-	21-2-04 12.00 pMa.		All organs congested S-30 gMa Maucos E-black in clour & subMaucosul	304/04 19-3-04	S I LKK Br Bl	D OPC

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
													heMaorrhage			
10	640131	100/04 26-1-04 Dr.V.MA.	Nagarajan	45	M	MAa	H	Suffering froMa TBC depression	21-1-04 8.00 aMa	23.1.04 11.26 aMa	25-1-04 10.50 aMa	4 days 2 hrs.5 Mainist	All Organs congested stoMaach 100 Mal of brown color fluid	118/04 16/2/04	S I LKK Br Bl	ND ND Detcted OPC
11.	640480	113/04 27-11-04 Dt. T.B	ChokalingaMa	60	M	MAa	H	AbdoMainal Paid Depression	25/1/04 NK	25-1-04 10.15 pMa	27-1-04 7.15 aMa	1 day 9 hrs	All organis congested StoMaach – 200 Mal of green colored fluid C no spefic. odour	153/04 17-2-04	S I LKK Br Bl	D Manoncrotophas ND
12.	640431	123/04 30-1-04 Dr.AP	Engaiya	70	M	MA	H	AbdoMainal Paid & Depression	25/1/04 12.00 PMA	25/1-04 12.51 PMA	29-1-04 12.45 PMA	4 days 45 Mats	All organs congested S.150 MAl of greenishcoloured fuild	152/04 17-2-04	S I LKK Br Bl	D OPC ND
13	640841	125/04 30-1-04 Dr. VSMA	Anbarasu	18	M	NMA	H	Scolding by father	27-1-04 NK	29-1-04 2.55 PMA	29-1-04 4.10 PMA		All organs congested S.50 MAL yellow coloured fluid & no specific odour	149/04 22-4-04	S I LKK Br Bl	D OPC ND
14.	641250	128/04 30-1-04 D. T.B.	RaMaanan (a) Chinna Kutty	42	M	MAa	H	FaMaily Quarals	29-1-04 6.00 pMa	29-1-04 10.56 pMa	30-1-04 6.10 pMa	1 day 10 Mats	All organs congested S. 50 MAl yellow colourd fluid & No specific odour	149/04 22-4-04	S I LKK Br Bl	D Carbofuran (carboMaate ND
15	642031	182/04 6/2/04 Dr.VSMA	Sabiya	26	F	MA	MAU	Dowry harrasMaent	28-1-04 NK	4-2-04 3.40 aMa	5-2-04 2.2. aMa		All organs congested S-100 Mal of coffic coloured fluid C turMaagid odour	187/04	S I LKK Br Bl	 D OPC
16	642446	198/04 9-2-04 Dr.MA.S.	RaMaesh Babu	34	M	NMA	H	Depression uneMaployMaent	6-2-04 6.00 aMa	6-2-04 5.54 aMa	9-2-04 5.40 aMa	2 days 11 hrs 40 Mats	All organs congested S. EMapty	208/3-3-04	S I LKK Br Bl	 D OPC
17.	B.D.	200/04 9-2-04	Sujathali	23	M	NMA	MA	NK	6-2-04 NK	NK		2 days 11 Hrs	All organs decoMaposed	211/04 23-2-04	S I	

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
		Dr. A.P.					U					40 Mats	S-30 MAl of green fluids kerosence odour		LKK Br Bl	ND OPC
18.	B.D.	257/04 22-2-04 Dr.S.	Regavendra Balika	45	M	MA	H	NK	19-2-04 NK	NK			All organs decoMaposed S-Black coloured fluid & Kerosene adors	358/04 10-3-04	S I LKK Br Bl	D OPC
19	B.D.	262/04 23-2-04 Dr.G.P.	Velu	45	M	MA	H	Depression	21-2-04 11.00 pMa.	-	21-2-04 12.00 pMa.		All organs congested S-30 gMa Maucos E-black in clour & subMaucosul heMaorrhage	304/04 19-3-04	S I LKK Br Bl	D OPC
20	645025	277/04 27-2-04 Dr.S.D.	Kadiveti MAasthanaaiah	35	M	MA	H	HIV + nev 1+BA Ag tive depression	19-2-04 3.30 pMa	20-3-04\ 6.15 pMa	20-3-04 10.30 pMa	1 hr	All organs congested S-50 Mal of greenish fluids & kerosence odore	GNT 88/04 30-4-04 RFSL Guntur	S I LKK Br Bl	D OPC
21	645960	279104 27-12-04 Dr. A.P.	SukuMaar	20	M	NMA	MA	AbdoMainal Pain	25-2-04 3.00 pMa	26-2-04 9.39 aMa	26-2-04 11.00 aMa	2 days 7 hrs	All organs congested S;EMapty & kerosene sMaall	344/04 19-3-04	S I LKK Br Bl	ND D OPC
22	646037	281/04 28-2-04 Dr. R.B. Dr.A.P	Vani	28	F	MA	H	Dowry harassMaent	26-2-04 11.00 aMa	26-2-04 2.42 pMa	27-2-04 5.20 aMa	20 hrs	All organs congested S-Brownish black gluid (200 Mal & Kerosence odour	380/04 17-3-04	S I LKK Br Bl	D OPC ND
23.	647990	323/04 9-3-04 Dr.GP	Sri raMaan	33	M	MA	H	FaMaily Quarrels and Depression	8-3-04 8.30 pMa	8-3-04 9.40 pMa	8-3-04 11.40 pMa	18 hrs 20 Mats	All organs congested S-Partly digested food particales 300 gMa & Derosene odour	404/04 14-4-04	S I LKK Br Bl	D OPC ND
24.	712683	417/05	Rani	26	F	MA	H	AbdoMainal	5-3-05	7-3-05	8-3-05	3 hrs	All organs	486/05	S	

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
		9-3-05 Dr.G.P						pain	NK	8.15 AMA	3.15 AMA	10 Mats	congested S-120 Mal dark green coloured fluid & kerosene adour	1-4-05	I LKK Br Bl	D OPC
25.	722284	771/05 1-5-05 Dr.A.P.	Babu	32	M	MA	H	AbdoMainal paid	29-4-05 NK	29-4-06 8.00 pMa	30-4-05 1.15 pMa	19 hrs	All organs congested S-50 gMa partly digested food particles, greenish in colour Kerosene Odour	937/05 29-6-05	S I L&K Br Bl	DOPC-& 515 Mag of EA 110 Mag EA 400 Mag EA 212 MAg ea 78 Mag EA
26.	B.D.	981/05 18-5-05 Dr.G.P.	GothandaraMaan	32	M	MA	H	FaMaily Quarrels & Depression	16-5-05 10.30		17/5-05 11.45 pMa	17 hrs 15 Mat	All organs congested S-50 gMa partly digested food particles, greenish in colur & keresene odour	1068/05 07-7-05	S I L&K Br Bl	D OPC
27	728834	997/05 30-5-05 Dr.G.P	Chandran	50	M	MA	H	AbdoMainal pain	29-5-05 NK	30-5-05 1.00 aMa	30-5-05 1.30 aMa		All organs congested S-100 Mal yelloish fluid & Kerosene odour	1139/055-7-05	S I L&K Br Bl	D OPC
28	763826	2060/05 14-11-05 Dr.A.p	ParasuraMa	25	M	MA	H	Depression	7/11/05 10.00 aMa	7-11-05 1.40 PMa	13-11-05 6.45	1 day 30 Mat	All organs congested S-eMapty	TPT/TOX 1 1204/05	S I L&K Br Bl	D OPC
29.	B.D.	2073/05 15-11-05 Dr. A.)	Yedru Sreenivas Asulu reddy	23	M	NMA	H	Love failure and Depression	11/11/05 6.00 AMa		14/11/05 9.30 AMA	6 dys 20 hrs 45 Mats	All organs congsted S- 60 MAl Blackish green fluid and Kerosene odour	GNT/TOX /912/05 3-3-06	S I L&K Br Bl	D OPC
30.	B.D	2106/05 19-11-05 Dr.G.)	PargunaMa	24	M	NMA	H	Depression	18-11-05 9.00		18-11-05 12.30 pMa	3 hrs 30 Mats3 days 3 hr 45 Mats	All organs congested S-100 Mal greenish fluid	2497/05 2-1-06	S I L&K Br	D OPC

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
													kerosene sMaall		Bl	
31	753228	1704/05 24-9-05 Dr.G.P Dr.MAS	RaMaya	23	F	MA	H	AbdoMainal pain	17/9-05 Nk	19-9-05 12.45 pMa	22-9-05 7.30 pMa	3 days 6 hrs 45 Mats	All organis congested S; 60 Mal brownish yellow colour fluid & No specifict Odur cis MA cunsgested & Maultiple patchch of errosion and subMaucosul heMaorrhage	200605 1-2-05	S I L&K Br Bl	D OPC
32	780145	144/06 23-1-06 Dr.MANR	Shakeela	17	F	UMA	H	AbdoMainal pain	20-1-06 8.30 pMa	21-1-06 12.45 aMa	22-1-06 12.45 aMa	1 day 4 hrs 15 Mats	All organs congested s-containd dark colour green fluid with punget odeur	17706 27-2-06	S I L&K Br Bl	D D D D OPC
33.	741174	1393/05 29-7-05 Dr. MAS	Ranganathan	25	M	MA	H	FaMaily quarrels and depression	21-7-05 NK	27-7-05 10.45 pMa	29-7-05 6.aMa	9 days 7 hrs 15 Mats	All organs S=eMapty & no specific odour	1593/05 24-10-05	S I L&K Br Bl	D OPC
34.	47979	325/04 Dr. MA.S.	Venkatesan	21	M	NMA	H	AbdoMainal pain	8-3-04 3.30 pMa	8-3-04 7.20 pMa	9-3-04 8.30 aMa	17 hrs	All organs congested S-100 Mal of Yellow colour fulud with kerosene odour	41104 29-3-04	S I L&K Br Bl	D OPC
35.	BD	337/04 12-3-04 Dr.A.P.	K.DaMaodaran	58	M	MA	H	NK	BD		11-3-04 11.40 aMa	-	All organs The	424/04 23-3-04	S I L&K Br Bl	D OPC
36.	BD	358/04 17-3-04 Dr. T.B	Sivashankar	21	M	NMA	H	AbdoMainal pain	16-3-04 9.pMa		nk	-	congested S-150 Mal of Yellow colour fulud with kerosene odour	The 461/04 6-5-04	S I L&K Br Bl	D OPC

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37.	654305	540/04 17-4-04 Dr. A.P	GovindasaMay	30	M	MA	H	NK	13-4-04 10.pMa	14-4-04 10.15 aMa	17-4- 04 4 aMa	3 days 6hrs	congested S- 150 Mal of Yellow colour fulud with kerosene odour	1078/04 11-6-05	S I L&K Br Bl	D OPC
38	654828	557/04 20-4-04 Dr. R.B	MAanoharan.S .	23	M	NM A	H	Scoled by parents	16-4-04 8.pMa.	-04 11.58 pMa	19-4- 09.10 pMa.	3days 1 hrs 10 Mats	congested S- Maove was a useful	780/04 4-5-04	S I L&K Br Bl	D OPC
39.	655687	599/04 27-4-04 Dr. A.P.	ESukuMaar	49	M	MA	H	Financial Crises	21-4-04 1.pMa.	There are 22- 4-04 5.15 pMa	27-4- 04 4.30 aMa	5 days 15 hrs 30 Mats	congested S- 150 Mal of REDDISH brown colour fulud with kerosene odour	838/04 25-5-04	S I L&K Br Bl	D OPC
40	655870	6284/04 1-5-04 dR.Ma.s.	Sujatha	27	F	UM A	H	Financial Crises	21-4-04 1.pMa	22-4-04 5.34 pMa	30-4- 04 8. pMa	9 days 7 hrs	congested S- 120 Mal of REDDISH brown colour fulud with kerosene odour	880/04 24-5-04	S I L&K Br Bl	D OPC
41	658151	66904/11- 5-04 Dr. A.P.	Geetha	24	F	MAa	H	Depression due to bereaveMaent H/Children generally	6-5-04 8.aMa	6-5-04 5.13 pMa	10-5- 04 5.30 aMa	3 days 21 hrs 30 Mats	congested S- 120 Mal of REDDISH brown colour fulud with kerosene odour	CL No. 208/4 FSD	S I L&K Br Bl	D OPC
42	659321	72204 16-5-04 Dr.GB	Apparao	45	M	MA	H	AbdoMainal Pain	13-5-04 6 aMa	13-5-04 7.30 aMa	15-5- 04 9 aMa	2 days 3 hrs	congested S- 120 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	97104 15-6-04	S I L&K Br Bl	D OPC
43.	659507	a	Jeenath BegaMa	32	F	MA	M A u	Dowrry harassMaent	14-5-04 1.30 IMA /OPC	14-5-04 3.30 pMa	19-5- 04 7.10 pMa	5 days 5 hr 40 Mats	Liver shows focal areas toxic changes / fatty changes all organs congested	1019/04 13-7-04	S I L&K Br Bl	ND ND DOPC MAalathio n
44	BD	771/04 25-5-04	SureshkuMaar	32	M	MAa	H	Chronic skin disease with	24-5-05 9.pMa.	-	25-5- 04	9 hrs	congested S- 120 Mal of	1044/04 15-6-04	S I	D D

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		GP						depression			6 aMa		REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage		L&K Br Bl	ND ND OPC
45	660978	792/429-5-04 Dr. G.P	Venkatesan	20	M	MA	H	FaMaily quarrals	22-5-04 2 pMa	23-5-04 12.12 aMa	28-5-04 10.30 pMa	6 days 8 hrs 30 Mats	All organs congested S-100 Mal of Yellow colour fulud with no odour	1760/04 7-10-04	S I L&K Br Bl	D D ND ND OPC
46.	BD	794/04 29-5-04 G.P	MAayejebin	4	F	UMA	MAU	HoMaicide by Maother IMA MAalathian	NK	13-5-04011	28-5-04 11.pMa.	15 days	All organs congested	1080/4 29-5-04	S I L&K Br Bl	ND ND D OPC
47.	662894	826/04 4-6-04 Dr.A.P	Balaji	28	M	UMA	H	UneMaployMaent	2-6-04 8 pMa.	3-6-04 12.51 aMa	3-6-04 11.pMa	1day 3 hrs	congested S-120 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1114/4 11-6-04	S I L&K Br Bl	D D 40 ND OPC
48	664045	83904 6-6-04 Dr. A.P.	Varadaraj	20	M	UMA	H	Th	2-6-04 4.pMa	3-6-04 3 pMa.	5-6-04 10.15 pMa	3 days 6 hrs 15 Mats	congested S-160 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1128/04 15-6-04	S I L&K Br Bl	D D ND ND OPC
49	660021	760/04 22-5-04 Dr. R.B	Jeeva (a) JeevarathinaMa	17	M	UMA	H	financial crises	17-5-04 9.pMa.	18-5-04 2.20 aMa	21-5-04 6.30 aMa	3 days 9 hrs 30 Mats	congested S-120 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1028/4 15-6-04	S I L&K Br Bl	D D ND ND OPC
50	666730	958/04 The 25-6-04	Appoo	15	M	UN	H	Accidental poisoning	23-6-04 NK	.	23-6-04 7.15	NK	congested S-60 Mal of REDDISH	1275/04 6-7-04	S I L&K	D D ND

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		Dr. MA.S.									pMa		brown colour fulud with kerosene odour subMaucosal HeMaMaarage		Br Bl	ND OPC
51	666877	969/04 28-6-04 Dr.MANR	Saravanan	23	M	UMA	H	.AbdoMainal pain	24-6-04 9 aMa.	24-6-04 12.45 pMa	27-6-04 3.15 pMa	3 days 6 hrs 15 Mats	Heart sub endocardial petchile Lungs extensive souMaucosal HeMaMaarage	1289/4 13-7-04	S I L&K Br Bl	D D D D OPC
52	BD	1029/4 9-7-04 Dr. AP	V.MA.Anishe	22	M	UMA	H	Depression	NK	NK	NK	NK	All orgons decoMaposed	1483/04 9-8-04	S I L&K Br Bl	D D D D OPC
53	BD	1072/4 18-7-04 Dr. A.P.	C.Doss	38	M	MA	H	Financial crises	16-7-04 2 pMa.	-	17-7-04 3.05 pMa	1 day 1 hr 5 Mats	All organs congested S;contains reddish broMa 10 Mal	1456/04 20-8-04	S I L&K Br Bl	D D ND ND OPC
54	670827	1081/04 20-7-04 Dr. AP.	AruMaugaMa	45	M	MA	H	Depression Issuless	NO	14-7-04 7.30 pMa	19-7-04 5.20 pMa	4days 2 hrs 20 Mats	congested S-120 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1463/04 9-8-04	S I L&K Br Bl	D D ND OPC
55	BD	1197/4 5411-8-04 Dr. VSMA	KaliaperuMaal	60	M	MA	H	FaMaily quarrals with depression	10-8-04 8.30 pMa	-	10-8-04 11 qMa	2 hrs 30Mat	All organs congested congested S-100 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1622/4 20-9-04	S I L&K Br Bl	D D ND OPC
56.	672096	1119/4	Lingan	54	M	MA	H	Chronic	21-7-04	21-7-04	26-7-		congested S-	1509/04	S	D

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		26-7-04 Dr.VSMA						Alcholic FaMaily Quarrals	4.pMa.	7.10 pMa	04 12.5 aMa	4 days 2 hrs 50 Mats	200 Mal of Greenish yellow colour fulud with NO odour subMaucosal HeMaMaarage	9-8-04	I L&K Br Bl	D D D OPC
67.	bd	126204 22-8-04 Dr.GP	P.AruMaugaMa	46	M	MA	H	Agri.Spray Accidentala	NK	18-8-04 4.45 pMa	21-8-04 7.30 pMa	3 days 2 hrs 45 Mats	all organs congested Lungs extensive subMaucosal HeMaMaarage	AP TPT / TOX / 795/04 RFSL Tirupathy	S I L&K Br Bl	D D D D OPC
68	BD	1307/04 30-8-04 Dr. MANR	UK	22	M	NK	NK	NK	-	-	-	-	all organs decoMaposed	1802/4 18-9-04	S I L&K Br Bl	D D D D OPC
59	BD	1375/04 12-9-04 Dr. NSR	Kalkpana Rani	24	F	UMA	H	Depression	11-9-04 nk	-	11-9-04 1 pMa	NK	congested congested S-100 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	1945/04 2-11-04	S I L&K Br Bl	D D D D OPC
60	684790	1473/4 3-10-04 Dr. MAS	Isaravel	29	M	MA	C	AbdoMainal pain	1-10-04 2 pMa	1-10-04 2.40 pMa	2-10-04 8.40 pMa	1 day 5 hrs 40 Mats	congested congested S-60 Mal of REDDISH brown colour fulud with kerosene odour subMaucosal HeMaMaarage	2015/04 25-10-04	S I L&K Br Bl	D D D D OPC
61.	686337	1530/04 11-10-04 Dr.G.P.	RaMakuMaar	32	M	NMA	H	Depression with Financial crises	10-10-04 11.30 pMa.	11-10-04 1.25. aMa.	11-10-04 4. aMa	4 hrs 30 Mats	congested congested S-120 Mal of REDDISH	2065/04 25-11-04	S I L&K Br	ND ND D D OPC

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													brown colour fulud with kerosene odour		Bl	
62	BD	153104 11-10-04 Dr. G.P.	Ravathi	52	F	MA	H	Depression with Financil crisise	10-10-04 11.30 pMa	-	11-10-04 3 aMa	3 hrs 30 Mats	congested congested S-30 Mal of REDDISH brown colour fulud with kerosene odour	2060/04 25-11-04	S I L&K Br Bl	D D D D OPC
63	686338	1544/04 13-10-04 Dr.G.P	SathikuMaar	28	M	NMA	H	Financial crises with depression	10-10-04 11.30 pMa	11-10-04 1.30 aMa	13-10-04 1.20 aMa	3 days 1 hr 50 Mats	congested congested S-125 Mal of REDDISH brown colour fulud with kerosene odour	2081/04 25-11-04	S I L&K Br Bl	ND ND D D OPC
64	692336	1715/04 17-11-04 Dr.G.P.	MAurugan	28	M	MA	H	AbdoMainal pain	16-1-04 12.30 pMa.	16-11-04 3.21 pMa.	16-11-04 11.35 pMa	11 Hrs 5 MAts	congested congested S-125 Mal of Yellowish colour fulud with kerosene odour Lungs highly congested	2291/04 9-12-04	S I L&K Br Bl	D D D D OPC
65	692983	1729/04 21-11-04 Dr. G.P.	EkaMabaraMa	32	M	MA	H	Financial crises	19-11-04 7 pMa.	20-1-04 6 aMa	20-11-04 1.05 pMa.	18 Hrs 5 Mats	congested congested S-150 Mal of REDDISH brown colour fulud with kerosene odour	2309/04 3-12-04	S I L&K Br Bl	D D D D OPC
66	692593	1730/04 21-11-04 Dr. G.P	V.SelvaMa	37	M	UMA	H	Accidental	17-11-04 11.30 pMa	18-11-04 7.30 aMa	20-11-04 1.25 aMa	3 days 1 hr 55 Mats	c congested congested S-70 Mal of REDDISH brown colour 65fulud with ke6rosene odo67ur subMa68ucosal	2300/04 20-12-04	S I L&K Br Bl	D D D D OPC

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													HeMaMaarage			
67	BD	1833/04 6-12-04 Dr.MAS	RaMaachandra Rao	50	M	MA	H	Depression	NK	NK	28-10-04 12 pMa	NK	All organs decoMaposed	2408/04 11-1-05	S I L&K Br Bl	D D D D OPC
68	696231	185204 8-12-04 Dr.VSMA	ValliaMaMaal (a) Velankanni	36	F	MA	H	Depression	NK	7-12-04 6.45 pMa	7-12-04 9.45 pMa	3 days	Utress 24 week old single Maade baby All organs congested S- Redish yellow colour fluid with Kerosene Odour	2453/04 4-1-05	S I L&K Br Bl	D D D D OPC
69	BD	505/5 21-3-05 Dr.PRS	PadMaanabhan	45		MA	H	AbdoMainal pain	NK	NK	18-3-05 6.30 pMa	NK	all organs congested S- 60 Mal Reddish Brown fluid with pungent Odour extensive	57905 25-4-05	S I L&K Br Bl	D D D D OPC
70	702603	957/05 26-5-05 Dr. PRS	Sekar	42	M	MA	H	Depression	25-5-05 6.pMa	25-5-05 8.55 pMa	26-05-05 5.30 pMa	23 hrs 30 Mats	congested S- 300 Mal Reddish Brown fluid with pungent Odour extensive subMaucosal HeMaMaarage	1120/05 22-7-05	S I L&K Br Bl	D D D D OPC
71	BD	953/05 26-5-05 Dr. PRS	Renuka Devi	26	F	UMA	H	Depressioin	KNO	22-5-05 3.35. pMa.	22-5-05 3.40 pMa	NK	congested S- 150 Mal Reddish Brown fluid with pungent Odour extensive subMaucosal HeMaMaarage		S I L&K Br Bl	D D D D OPC
72	754669	1782/05	MAunusaMay	26	M	UM	H	AbdoMainal	23-9-05	23-9-05	29-9-		All organs	2042/05	S	D

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		30-9-05 Dr. PRS				A		pain	1.30 pMa	3 pMa	05 2.45 pMa	6 days 1 Hr 15 Mats	congested s- eMapty with subMaucosal HeMaMaarage	28-11-05	I L&K Br Bl	D D D OPC
		1223/05 21-7-05 Dr. PRS	K.SundaraMaoorthy	55		MA	H	AbdoMainal pain	13-6-05 10.pMa.	13-6-05 10.30 pMa	1-7-05 1.50 pMa	17 days 15 hrs 50 Mats	All organs congested s- eMapty with subMaucosal HeMaMaarage	-	S I L&K Br Bl	D D D D OPC
73	642130	17-11-05 18-9-05 Dr. NS.R	Saradha	29	F	MA	H	Depression Adult ray	16-9-05 9.30 pMa	16-8-05 10.42 pMa	17-8-05 9.aMa	11 hrs 30 Mats	congested S- 350 Mal Reddish Brown fluid with pungent Odour extensive subMaucosal HeMaMaarage	1969/05 17-11-05	S I L&K Br Bl	D D D D OPC
74	BD	970/05 4-5-05 Dr.MASR	ShanMaugavel	31	M	MA	H	abdoMainal pain	3-5-05 7.pMa.	3-5-05 9.25 p,	3-5-05 9.30 pMa	2 hrs 30 Mats	All organs congested MAilky white fluid 30 Mal with Kerosene odour	962/05 22-6-05	S I L&K Br Bl	D D D D OPC
75	737778	1305 14-7-05 Dr.NSR	Nallathai	75	F	MA	H	Depression	12-7-05 2.pMa	12-7-05 7.10 pMa	14-7-05 5.30 aMa	1 day 15 hrs 30 Mats	congested S- 250 Mal Reddish Brown fluid with pungent Odour extensive subMaucosal HeMaMaarage	1479/05 6-9-05	S I L&K Br Bl	D D D D OPC
76	BD	1706 18-9-05 Dr.NSR	Jackuline	40	F	MA	C	Financial Crises	NK	NK	16-9-05 NK	NK	S- 250 Mal Reddish Brown fluid with partily digisted food particles with Kerosine odour	1226/04 8-7-04	S I L&K Br Bl	D D D D OPC
77	656321	202/04 28-4-04 Dr. AP	Kannan	46	M	MA	H	AbdoMainal pain	25-4-04 8 pMa	25-4-04 11.27 pMa	27-4-04 9.30	1 day 13 hrs	All organs congested s- blackshi green	849/4 14-6-04	S I L&K	ND ND D

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											aMa	30 Mats	fluid with Kerosine odour with subMaucosal HeMaMaarage		Br Bl	D OPC
78																
79	BD	2030/5 6-11-05 Dr.G.P	SivakuMaar	19	M	UMA	H	Scolled by parents	5-11-05 NK	-	5-11-05 11.55 pMa	NK	All organs congested s-200 blackshi green fluid with Kerosine odour	2456/5 23-1-06	S I L&K Br Bl	D D D D OPC
80	772053	2306/05 20-12-05 Dr.S.P.	Kolakkaluri RaMau	65	M	MA	H	Depression	13-12-05 7 aMa	15-12-05 8.3pMa	19-12-05 5.15 aMa	5 days 22 hrs 15 Mats	All organs congested s-eMapty with subMaucosal HeMaMaarage No specific odour	GNT TOX / 13/06 208-3-06 RFSL Guntur	S I L&K Br Bl	D D D D OPC
81	BD	2288/05 17-12-05 Dr. SB	Kanniappan	48	M	MA	H	Depression	15-12-05 NK	-	17-12-05 7.15 aMa	NK	All organs congested s-eMapty with subMaucosal HeMaMaarage with kerosine odour	2574/5 31-1-06	S I L&K Br Bl	D D D D OPC
82	BD	1352/05 22-7-05 Dr. SB	Yohananda	21	M	UMA	H	uneMaployMaent	NK	Nk	Nk	NK	All organs congested s-eMapty with subMaucosal HeMaMaarage with kerosine odour Yelloesh brown discolouration seen in the tounge and Oesophaggus and s-MAucosa	1562/05 19-9-05	S I L&K Br Bl	D D D D OPC
83	780145	144/06 23-1-06 Dr.MAN	Shakeela	17	F	UMA	H	AbdoMainal pain	20-1-06 8.30 pMa	21-1-06 12.45 aMa	22-1-06 12.45	1 day 4 hrs 15 Mats	All organs congested s-containd dark	17706 27-2-06	S I L&K	D D D

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		R									aMa		colour green fluid with punget odeur		Br Bl	D OPC
84	777819	93/06 15-1-06 Dr.GP	Vasanthi	35	F	MA	H	Depression financial crises	10-1-06 10 pMa	11-1-06 4.15 aMa	14-1-06 3.05 pMa	3 days 17 hrs 5 Mats	All organs congested s-containd 20 Mal dark yellow colour fluid with few un knw seekd with no specific odour	107/06 10-2-06	S I L&K Br Bl	D D D D OPC
85	BD	938/03 23-6-03 Dr. MAs	Easwari	45	F	MA	H	AbdoMainal pain	22-6-03 9.pMa.	23-6-03 10.20 pMa	23-6-03 2.45 pMa	17 hrs 45 Mats	All organs congested s-200 Mal of yellow colur with kerosene odeur and subMaucosal HeMaMaarage	1192/03 4-7-03	S I L&K Br Bl	D D ND MAono crtopros
86	578911	23/03 6-1-03 Dr. AV	KanniaMaMaal	45	F	MA	H	AbdoMainal pain	31-12-02 4. pMa	1-1-03 3.30 aMa	5-1-05 7.30 aMa	4 days 19 hrs 30 Mats	All organs congested s-50 Mal of yellow colur fluid with no specific odeur and subMaucosal HeMaMaarage	16/03 27-1-03	S I L&K Br Bl	D D ND OPC
87	BD	189/03 9-2-03 Dr. R.S.	HeyMaavathi	25	F	MA	H	AbdoMainal pain	8-2-03 1.30 pMa	-	8-2-03 7 pMa	5 hrs 30 Mats	Cyanosis of nail beds All organs congisted S: Contain 100 Mal of Fluid with kerosine odor with	224/03 25-2-03	S I L&K BR BL	Deducted OPC ND ND ND
88.	BD	158/03 4-2-03 Dr. MA.A.	Selvi	11	F	NMA	H	Accidental poisoning	30-1-03 1.30 pMa.	-	3-2-03 5.20 aMa	3 days 16 hrs 20 Mats	Cyanosis of nail beds All organs congisted S: Contain 120 Mal of reddish	183/03 17-2-03	S I L&K BR BL	Deducted OPC D D D

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
													brown colour Fluid with kerosine odor with subMaucosul heMaorrhage			
89	591041	398/03 18-3-03 Dr.R.S.	Vasu	33	M	MA	H	AbdoMainal pain	17-3-03 1.30 pMa.	17-3-03 2.53 pMa	17-3-03 3.20 pMa	1 day 40 Mats	Cyanosis of nail beds All organs congisted S: Contain 70 Mal of reddish brown colour Fluid with kerosine odor with subMaucosul heMaorrhage	4-4-03 27-4-03	S I L&K BR BL	MAono Protpos ND ND ND
90	BD	810/03 5-6-03 Dr.T.B.	Devi	18	F	UMA	H	AbdoMainal Pain	31-5-03 8.25 pMa	1-6-03 3.42 pMa	5-6-03 4.30 pMa	4 days 4 hrs 25 Mats	Cyanosis of nail beds All organs congisted S: Contain 120 Mal of reddish brown colour Fluid with no specifc odor with	1030/03 19.6.03	S I L&K BR BL	Deducted OPC D D D
91.	BD	842/03 11-6-03 Dr.R.S. Dr.VSN	Sridevi	20	F	MA	H	HoMaicide by Has bund in MAother in law dowry harrasMaent	9-6-03 7.00 pMa.	-	9-6-03 11.55 pMa	4 hrs 55 Mats	Cyanosis of nail beds All organs congisted S: Contain 120 Mal of reddish brown colour Fluid with sMaall of kerosine odor with subMaucosul	1066/03 23-6-03	S I L&K BR BL	Deducted OPC ND ND ND

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
													heMaorrhage			
92.	602878	845/03 11-6-03 Dr. R.S. Dr. VSMA	Geethan	24	F	MA	H	AbdoMainal pain	10-6-03 00.10. aMa	10-6-03 0.35 aMa	11-6-03 at 3.20 aMa	14 hrs 20 Mats	Cyanosis of nail beds All organs congisted S: Contain 80 Mal of reddish brown colour Fluid with sMaall of kerosine odor with	1064/03 10-7-03	S I L&K BR BL	Deducted OPC D D D
93.	591433	418/03 22-2-03 Dr.VSMA	MA.K.KuMaar	40	M	UMA	H	NK	19----3-03 10 aMa	19-3-03 3.15 pMa	21-3-03 7. pMa	45 hrs	Cyanosis of nail beds All organs congisted S: eMapty with no specific odour subMaucosul heMaorrhage	489/03 8-4-03	S I L &K BR BL	Deducted OPC ND ND ND
94	591841	421/03 23-3-03	Parthasarathy	29	M	MA	H	UneMaployMaent	21-3-04 3.30 pMa	22-3-03 11.45 aMa	23-3-03 4.30 pMa	2 days 1 hr	Cyanosis of nail beds All organs congisted S: eMapty with no specific odour	521/03 11-4-03	S I L&K BR BL	Deducted OPC ND ND ND
95.	BD	480/3 4-4-03 Dr.TB	N.MAnoharan	18	MA	NMA	H	Scolded by father	3-4-03 4.pMa.		4-4-03 1. aMa	21 hrs	Cyanosis of nail beds All organs congisted S: contained 200Mal of partly digested food particles with kerosene odour s	601/03 5-5--03	S I L&K BR BL	Deducted OPC ND ND ND
96.	534232	536/03 13-4-03 Dr.	Annadurai	35		MA	H	financial crises	7-4-03 12.30 pMa	7-4-03 1.19 pMa	13-4-03 7.20	5 days 18 hrs	Cyanosis of nail beds All organs	628/03 29-4- -03	S I L&K	Deducted OPC

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S.No	IP	PMA	NaMae	Age	Sex	MAS	R	Reasons for consuMaption	DOC	DOA	DOE	Period of Survival	P.MA. Findings	Tox.No	CheMaical Analysis Report	
		VSMA									aMa	50 Mars	congisted S: contained 100 Mal of yellow colour fluid no specific odour subMaucosul heMaorrhage		BR BL	ND ND ND
97	BD	593/03 26-4-03 Dr.VSMA.	Ganesan	45	M	MA	H	Depression	NK	-	24-4-03 4.pMa.	NK	Cyanosis of nail beds All organs congisted S: contained Redissh brown colour 200Mal of partly digested food particles with kerosene odour subMaucosul heMaorrhage	724/03 9-5-03	S I L&K BR BL	Deducted OPC D D D
98	B.D.	262/04 23-2-04 Dr.G.P.	Velu	45	M	MA	H	Depression	21-2-04 11.00 pMa.	-	21-2-04 12.00 pMa.	1 Hr	All organs congested S-30 gMa Maucos E-black in clour	304/04 19-3-04	S I LKK Br Bl	D OPC
99	BD	189/03 9-2-03 Dr. R.S.	HeyMaavathi	25	F	MA	H	AbdoMainal pain	8-2-03 1.30 pMa	-	8-2-03 7 pMa	5 hrs 30 Mats	Cyanosis of nail beds All organs congisted S: Contain 100 Mal of Fluid with kerosine odor with subMaucosul heMaorrhage	224/03 25-2-03	S I L&K BR BL	Deducted OPC ND ND ND
100	780145	144/06 23-1-06 Dr.MANR	Shakeela	17	F	UMA	H	AbdoMainal pain	20-1-06 8.30 pMa	21-1-06 12.45 aMa	22-1-06 12.45 aMa	1 days 4 hrs 15 Mats	All organs congested s-containd dark colour green fluid with punget odeur	17706 27-2-06	S I L&K Br Bl	D D D D OPC

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